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IN THE CLAIMS:

Please cancel claims 10-32 and 42-59.

Please amend claims 1 and 33 in the following manner:

1	1. (Currently amended) A method for identifying a therapeutic agent for
2	use in treating a constitutive androstane receptor (CAR)-mediated disorder or condition,
3	the method comprising:
4	identifying a candidate therapeutic agent by screening one or more
5	compounds to determine whether said compounds can modulate a CAR-mediated
6	intermolecular interaction;
7	administering the candidate therapeutic agent to a test mammal; and
8	determining whether the level of a cholesterol indicator is modulated in
9	said test mammal.
1	2. (Original) The method of claim 1, wherein said candidate therapeutic
2	agent is 5β-pregnan-3,20-dione.
1	3. (Original) The method of claim 1, wherein said CAR-mediated
2	disorder or condition is selected from the group consisting of: hypercholesterolemia,
3	lipid disorders, atherosclerosis, and cardiovascular disorders.
1	4. (Original) The method of claim 1, wherein the mammal is a
2	cholesterol-elevated mammal.
1	5. (Original) The method of claim 4, wherein the test mammal has a
2	disruption in both CAR alleles.
1	6. (Original) The method of claim 1, wherein said cholesterol indicator is
2	the level of serum cholesterol.

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7. (Original) The method of claim 1, wherein said cholesterol indicator is 1 the level of a member selected from the group consisting of HDL cholesterol, LDL 2 cholesterol, and VLDL cholesterol. 3 8. (Original) The method of claim 1, wherein said cholesterol indicator is 1 the mRNA level of a gene involved in the regulation of cholesterol levels. 2 9. (Original) The method of claim 1, wherein said CAR-mediated 1 intermolecular interaction is CAR-mediated gene expression. 2 10-32. Cancelled. 33. (Currently amended) A method for identifying a therapeutic agent for 1 use in treating a constitutive androstane receptor (CAR)-mediated disorder or condition 2 the method comprising: 3 administering a compound to a CAR compromised mammal; and 4 determining whether administration of the compound results in a change 5 in cholesterol level compared to a mammal to which the compound is not administered. 6 34. (Original) The method of claim 33, wherein the method further 1 comprises administering the compound to a CAR non-compromised mammal and 2 comparing the effect on the cholesterol level indicator of administering the compound to 3 that of administering the compound to the CAR compromised mammal. 4 35. (Original) The method of claim 33, wherein said cholesterol level 1 2 indicator is the level of serum cholesterol. 36. (Original) The method of claim 33, wherein said cholesterol level 1 2 indicator is the level of a member selected from the group consisting of HDL cholesterol, 3 LDL cholesterol, and VLDL cholesterol. 1 37. (Original) The method of claim 33, wherein said cholesterol level indicator is the mRNA level of a gene involved in the regulation of cholesterol levels. 2

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1	38. (Original) The method of claim 33, wherein said CAR compromised
2	mammal is a mammal having a disruption in both CAR alleles.
1	39. (Original) The method of claim 38, wherein said CAR compromised
2	mammal is a mouse.
1	40. (Original) The method of claim 38, wherein said disruption occurs in
2	the coding region for the DNA binding domain of CAR.
1	41. (Original) The method of claim 38, wherein said disruption in a CAR
2	allele comprises an insertion at codons for amino acid positions from about amino acid 21
3	to about amino acid 86 of CARβ.

42-59. Cancelled.